Plant Breeding **120**, 169—172 (2001)

ISSN 0179-9541

Inheritance of resistance to early blight disease in a diploid potato population

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With 1 figure and 1 table

Received December 2, 1999/Accepted July 11, 2000
Communicated by C. O. Qualset

Abstract

Early blight disease, caused by *Alternaria solani* Sorauer, is a serious disease of potato foliage and tubers that occurs in most potato-growing regions world-wide. Developing new potato cultivars with resistance to early blight may reduce losses in the field and in storage, and lessen the need for fungicide applications. A total of 280 clones, derived from 72 maternal half-sib families from a diploid random-mated hybrid population of *Solanum phureja* × *Solanum stenotomum* were examined for resistance to early blight. The clones that were evaluated in a replicated field trial for 2 years in Pennsylvania, USA, had similar early blight intensity both years. Significant differences were found among families, within families and for the interaction of years × within families. Broad-sense heritability for resistance, measured as area under the disease progress curve (AUDPC), was estimated as 0.73, with a 95% confidence interval of 0.65–0.78, and narrow-sense heritability was estimated as 0.61 ± 0.29 ($P = 0.05$). The correlation of AUDPC for early blight between years was 0.57 ($P < 0.0001$). These results suggest that this diploid population is worthy of use in breeding for early blight resistance.

Key words: *Solanum phureja* — *Solanum stenotomum* — *Alternaria solani* — broad-sense heritability — narrow-sense heritability

Early blight, caused by *Alternaria solani* Sorauer, is a serious disease of potatoes that occurs in most potato-growing regions world-wide. Pelletier and Fry (1990) and Christ (1990) have reported that early blight has increased in severity in recent years owing to a combination of factors: the more widespread production of susceptible cultivars; reduced use of protectant fungicides associated with the adoption of spray-forecasting models for control of late blight, caused by *Phytophthora infestans* (Mont.) de Bary; and the use of fungicides for late blight control that are not particularly effective for early blight control (Nutter and MacHardy 1981).

Both foliage and tubers can become infected, although relatively little research has been done on the tuber-rot phase of this disease (Pavek and Corsini 1994). In foliage, early blight appears as target-like leaf spots on older, often senescing foliage. As the disease progresses, these spots may expand, eventually coalesce and, in severe cases, leaves drop off the plant. Young plants are resistant to early blight but older, more mature plants are much more susceptible (Pelletier and Fry 1989, 1990), and early-maturing cultivars are more susceptible to early blight than later-maturing cultivars (Harrison et al. 1965, Douglas and Pavek 1972, Holley et al. 1983). Yield losses of 20–30% caused by early blight have been reported (Johnson et al. 1986, Pelletier 1988).

Potato germplasm has been evaluated for resistance to early blight by a number of researchers (LeClerg 1946, O'Brien and Akeley 1971, Douglas and Pavek 1972, Bussey and Stevenson 1991, Christ 1991). Sources of resistance in *Solanum tuberosum* L. are relatively rare (Shtienberg et al. 1995). However, Platt and Reddin (1994) have identified some cultivars and advanced breeding selections with moderate resistance. Unfortunately, there has been little use of this germplasm specifically for developing cultivars with resistance to early blight (Pavek and Corsini 1994). This is probably because commercial cultivars with resistance are usually low yielding and late maturing (Rotem 1994). In an extensive evaluation of 934 potato clones from breeding programmes around the world, Boiteux et al. (1995) found a strong correlation between early blight resistance and late maturity, although some clones were found to be both resistant and earlier-maturing than the rest. The development of early blight-resistant cultivars can be expected to increase profitability by reducing the amount of fungicides used to produce a crop (Christ 1990, Stevenson 1994).

Both additive and non-additive genetic effects have been reported for early blight resistance in tetraploid potatoes (Brandolini 1992, Gopal 1998). However, additive genetic effects appear to predominate in the diploid potato species studied to date (Herriott et al. 1986, Ortiz et al. 1993), in 4*x*–2*x* hybrids involving these diploids (Herriott et al. 1990), and in the first backcrosses of the tetraploids derived into *S. tuberosum* (Christ and Haynes 1997). Herriott et al. (1986) and Ortiz et al. (1993) reported that resistance to early blight could be found in a diploid hybrid population of *Solanum phureja* × *Solanum stenotomum*. Herriott et al. (1986) estimated narrow-sense heritability for early blight resistance in this population using offspring–midparent regression as 0.83. Ortiz et al. (1993) also estimated narrow-sense heritability, which ranged from 0.64 to 0.78, for early blight resistance using parental materials from this same population in crosses with haploids, species and haploid–species hybrids in nested and diallel mating designs. Herriott et al. (1990) subsequently transferred this resistance to tetraploid *S. tuberosum* via 4*x*–2*x* crosses. Haynes and Christ (1999) recently have evaluated this diploid hybrid *S. phureja* × *S. stenotomum* population, which has undergone several additional cycles of selection for high specific gravity (Haynes et al. 1995, Haynes 2000) since the study reported by Herriott et al. (1986), for resistance to late blight caused by *P. infestans*, another important disease of potato. They estimated narrow-sense heritability for late blight resistance as 0.78 ± 0.29.

Narrow-sense heritability, as a measure of the ratio of additive genetic variance to phenotypic variance, is important to estimate for breeding at the diploid level. Broad-sense heritability, as a measure of the ratio of genetic (additive plus dominance) variance to phenotypic variance, is important to estimate since portions of the dominance variance can be transferred from the diploid parent to the tetraploid offspring in 4*x*–2*x* crosses (Haynes 1990).

The purposes of this study were to estimate both broad-sense and narrow-sense heritability for early blight resistance in a diploid hybrid population of *S. phureja* × *S. stenotomum*.

Materials and Methods

A total of 280 potato clones derived from several cycles of random-mating and recurrent selection for high specific gravity (Haynes et al. 1995, Haynes 2000) within a diploid hybrid population of *S. phureja* × *S. stenotomum* were evaluated for their reaction to early blight, caused by *A. solani*. In a previous study, using this same population, Haynes and Christ (1999) investigated the inheritance of resistance to late blight. This diploid population was composed of four clones from each of 72 maternal half-sib families (one clone from each of eight families was missing). Each family can be traced through the maternal parent back to one of the original founding plant introductions used to establish the population in 1966 (Haynes 1972, Haynes 1980). *S. phureja* and *S. stenotomum* are diploid cultivated species represented by many cultivars ranging from Bolivia to Columbia. The main barrier to movement of genes from this germplasm source to cultivated *S. tuberosum* L. is short day-length adaptation, which makes hybrids and backcross generations low-yielding by virtue of late onset and slow progression of tuberization under long-day growing environments. Through recurrent mass selection Haynes (1972, 1980) adapted a hybrid diploid population of these two species to the long-day growing conditions in North Carolina. Beginning in 1986, the population was subjected to several cycles of recurrent selection for high specific gravity, as reported in previous studies (Haynes et al. 1995, Haynes 2000). This population is very important because it has a long history of preselection for adaptation and now is a good source of genes for specific traits.

In 1997 and 1998, this population was evaluated for resistance to early blight in field trials in Rock Springs, Pennsylvania, USA. Two-hundred and eighty diploid clones, plus the moderately susceptible control cultivar 'Atlantic', were planted on a Hagerstown silty clay loam soil in a randomized complete block design consisting of two replications, four hills per plot. Plants were spaced 22 cm within the row, with 5 m between plots and 0.9 m between rows. Tubers were planted on 29 May 1997 and on 21 May 1998.

Fertilizer was applied in furrows at the rate of 106.2 kg/h of 10–10–10 (N–P–K) in 1997 and 101.9 kg/h of 5–10–20 (N–P–K) in 1998. Standard commercial management practices were used throughout the season, except that no fungicides were applied. For both years, the previous crop was red clover, which was mowed and ploughed in. During the 1997 growing season irrigation was applied on 1 and 17 July. During the 1998 growing season irrigation was applied on 5 August.

Natural inoculum was used as the source for the epidemic. A highly susceptible cultivar, 'Norland' was planted adjacent to each treatment row to increase the inoculum level and ensure uniform inoculum to the plots. Per cent defoliation caused by the early blight fungus was estimated visually in each plot three times near the end of each growing season: 19 and 28 August and 5 September 1997 and 3, 11 and 18 August 1998.

Area under the disease progress curve (AUDPC) was calculated (Shaner and Finney 1977) and subsequently analysed using the general linear models procedure in SAS (SAS Institute 1987). Type III sums of squares were computed because there were eight missing plots. All effects were considered random. Estimates of the clonal variance (σ^2_C), year × clone variance (σ^2_{YC}), and the error variance (σ^2_e) were calculated using the mixed procedure in SAS (SAS Institute 1996). Broad-sense heritability (H) on a half-sib family mean basis was calculated from these estimates of variances as:

$$H = \sigma^2_C/(\sigma^2_C + \sigma^2_{YC}/r + \sigma^2_e/ry),$$

where r = number of replications and y = number of years (Nyquist 1991), and a 95% confidence interval on H was calculated from mean squares (Knapp et al. 1985). Clonal variance was partitioned into variance among families (σ^2_F), and variance within families (σ^2_{HSF}), also using the mixed procedure in SAS (SAS Institute 1996). In the absence of epistasis, the variance among families is equal to $\sigma^2_A/4$ and the variance of half-sibs within families is equal to $(3\sigma^2_A)/4 + \sigma^2_D$, where σ^2_A is the additive genetic variance and σ^2_D is the dominance variance. Variance components were used to estimate narrow-sense heritability (h^2) as:

$$h^2 = \sigma^2_A/(\sigma^2_C + \sigma^2_{YC}/r + \sigma^2_e/ry)$$

(Nyquist 1991). The standard error of h^2 can then be estimated as 4 SE $(\sigma^2_F)/\sigma^2_P$, where σ^2_P is the phenotypic variance (Hallauer and Miranda 1981).

To detect significant early blight resistance in this population, the mean AUDPC of each diploid clone was compared with the mean AUDPC of 'Atlantic' using a least significant difference at the 5% level.

Results

Early blight pressure was similar in both years of the study, as indicated by the lack of significant year effects on AUDPC (Table 1); the mean AUDPC in 1997 and 1998 was 342 and 318, respectively. However, there were significant rep(year) effects (Table 1), which accounts for the rather high coefficient of variation (CV = 46) obtained. Early blight disease started earlier and was more severe at one end of the field (one rep.) than the other (second rep.). Temperatures were moderate both years and the 1997 growing season had low precipitation during June (59 mm) and July (60 mm), but normal precipitation during August (171 mm). The 1998 growing season had

Table 1: Analysis of variance and estimates of the variance components on area under the disease progress curve for 280 clones (four half-sib individuals from each of 72 families)[1] from a diploid hybrid *Solanum phureja* × *S. stenotomum* population evaluated for per cent foliar early blight (*Alternaria solani*) in Pennsylvania, USA, in 1997 and 1998

	Analysis of variance		Variance components	
Source[2]	df	Mean squares	Estimate	Standard error
Year	1	161 051		
Rep (year)	2	123 462**		
Clone	279	155 835**	28 561**	3442
Family	71	240 308*	6048*	2891
HS (family)	208	126 275**	22 755**	3353
Year × clone	276	42 504**	9520**	1939
Year × family	71	54 896*	2034	1291
Year × HS (family)	205	38 254**	7508**	2031
Error	557	23 419		
Total	1115			

*,** Significant at P = 0.05 and P = 0.01, respectively.
[1] One clone was missing from each of eight different families.
[2] Family = among families; HS(family) = within families.

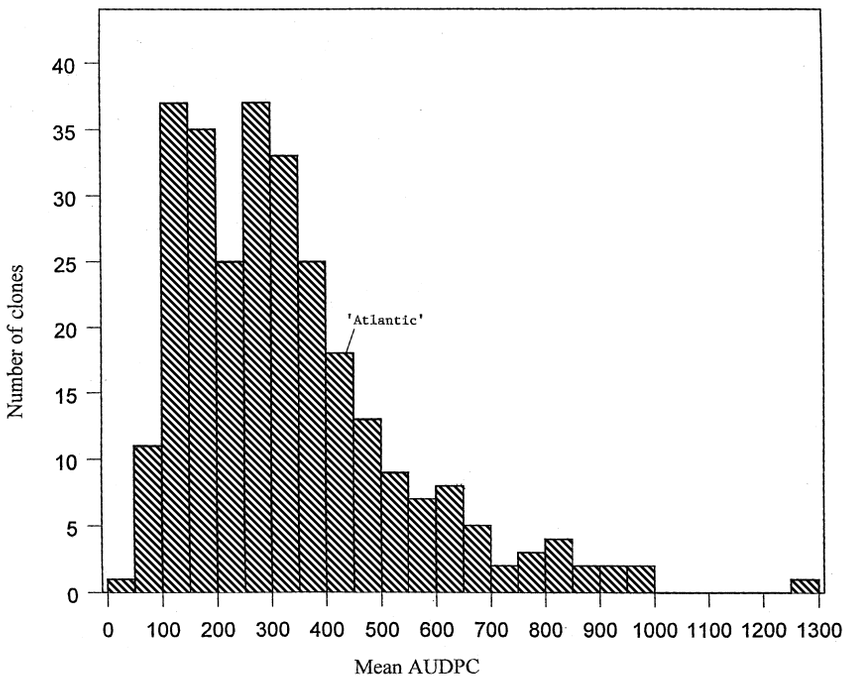

Fig. 1: Distribution by mean AUDPC of the 280 clones from a diploid hybrid *Solanum phureja* × *Solanum stenotomum* population evaluated for per cent foliar early blight in Pennsylvania, USA, in 1997 and 1998. Mean AUDPC of the control cultivar 'Atlantic' was 434

normal rainfall in June (131 mm) but low precipitation in July (89 mm) and August (71 mm).

The application of higher fertilizer and low rainfall in the early part of the season prevented an epidemic from starting until August in 1997. With the lower fertilizer application and higher rainfall early in the season in 1998, the epidemic started at a time more typical for the area. In 1998, the first symptoms were observed in the spreader rows of 'Norland' in early July with early blight spreading to the plots by late July. There was a significant linear correlation among clones for AUDPC between years ($r = 0.57$, $P < 0.002$).

There were significant differences among the clones (Table 1). Mean AUDPC over years for clones ranged from 46 to 1271 and the mean AUDPC for 'Atlantic' was 434. The distribution of mean AUDPC over the two years of testing for the 280 clones is shown in Fig. 1. Ninety-two clones had significantly lower mean AUDPC than 'Atlantic'.

The clone, year × clone and error variances were estimated as 28 561, 9520, and 23 419, respectively (Table 1). Broad-sense heritability on AUDPC for early blight resistance was estimated as 0.73 with a 95% confidence interval of 0.65–0.78. Upon further partitioning of the clonal source of variation, the variations among families and within families were also significant. In general, each family had a range of resistant to susceptible clones, however, all members of two families were resistant to early blight, whereas all members of three families were susceptible. The variation among families was only about one-quarter the variation within families (Table 1). Additive and dominance variances were estimated as 18 144 and 4611, respectively. Narrow-sense heritability on AUDPC for early blight resistance was estimated as 0.61 ± 0.29 ($P = 0.05$).

Discussion

In agreement with the findings of other researchers (Herriott et al. 1986, Ortiz et al. 1993) we have found that early blight resistance is highly heritable in this population and environment. Our estimate of narrow-sense heritability is lower than that obtained by Herriott et al. (1986), but their estimate was biased upwards because parents and offspring were evaluated in the same year. However, our estimate may also be biased upwards because the clones were evaluated at the same location in both years. The year–clone interaction accounted for only 12% of the total variation. Our estimate was obtained from evaluations of a population composed of 280 individual clones, each of which can be traced back to one of the original 72 founding plant introductions, not just a small sample of parents from that population. It is therefore more indicative of the potential of this population to contribute germplasm to the early blight breeding effort. Also, in agreement with other researchers (Holley et al. 1983, Pelletier and Fry 1989, 1990) the field resistance reported here affects the rate of development, and not the presence or absence of the disease. None of the clones was immune to infection by *A. solani*. There is no doubt the germplasm in this population is late-maturing. However, compared with earlier generations of selection for adaptation in this population, where tuberization was extremely low (Gautney 1983), this population now tuberizes readily, and the average yield per hill in 1995–96 in Maine, USA, was 413 g (Haynes 2000), compared with less than 75 g in North Carolina, USA, in the study by Gautney (1983). This increased yield may be the result of recurrent selection that has increased adaptation to long-day growing conditions, shorter maturity (although still considered late in maturity) than in earlier generations, and less stressful growing environments in Maine compared with North Carolina.

The estimates of heritability for resistance to early blight (0.61) and late blight (0.78) in this population are fairly large, indicating that additive genetic variance predominates. This suggests that genetic gains in resistance to either of these pathogens in this population can be readily achieved. With such large estimates of heritability, it might be suspected that relatively few genes are involved in resistance. Studies are currently underway using molecular techniques to determine

how many genes may be involved in resistance to either pathogen, and if any genes confer dual resistance. The correlation of resistance to early blight and late blight, as measured by AUDPC, were moderately low, ranging from 0.25 to 0.35 and explain only 6–12% of the variation. This would suggest that different genes are involved in resistance to these two pathogens in this potato population.

The development and enhancement of this particular diploid hybrid population over the past 30 years is influencing the USDA/ARS tetraploid cultivar breeding programme. In 1999, approximately 10% of the advanced selections evaluated as potential new cultivars are the result of either direct 4*x*–2*x* crosses or 4*x*–2*x* hybrids backcrossed once into *S. tuberosum*.

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